passed through the conducting airways and deposited on alveolar surfaces. Because silica crystals were translocated to macrophages and to Type I - epithelial cells, significantly fewer particles were present on alveolar surfaces 24 hrs post-exposure. At this time, high percentages of silica-containing macrophages were seen in the lavage and in situ, and these high levels of macrophage participation were maintained through 24 days post-exposure. Interestingly enough, macrophage populations were not perturbed beyond normal limits regarding cell number, viability, bacterial killing, O_2 consumption and enzyme release. There is ultrastructural evidence of macrophage deterioration, although the studies of lavaged populations were not sensitive enough to detect any changes. We propose that the events reported here are components of normal, steady-state clearance which is operative subsequent to inhalation of a sub-pathogenic dose of potentially toxic particulates.

DEPOSITION AND TRANSLOCATION OF INHALED SILICA

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Crystalline quartz (silica, SO_2) is a highly fibrogenic dust which causes significant lung disease in occupationally exposed individuals. While this has been known for many years, numerous questions exist regarding the early particle-cell interactions which lead to the initial lesions of silicosis. Virtually no information is available on the sites of initial particle deposition and the time-related events of silica translocation to various pulmonary cells. Such information is essential to achieving an understanding of the basic mechanisms of silica-induced lung disease.

White rats were exposed to 100 mg/m^3 of aerosolized alpha-quartz for 3 hrs in inhalation chambers. Groups of animals were sacrificed at the following times after exposure: 0 (immediately after exposure), 6, 12, 24, 48 and 72 hrs and 10, 14, 24 and 42 days. The lungs of these animals were fixed *in situ* for light and electron microscopy by vascular perfusion through the right ventricle. In addition, macrophages were recovered by bronchopulmonary lavage from groups of sham and silica-exposed animals at the time periods noted above.

Immediately after exposure, we found by quantitative backscatter electron imaging that there were approximately 17,000 silica particles per mm^2 of alveolar duct surface. Twenty-four hours after inhalation, only 3,000 particles/mm^2 remained on the duct surfaces. Transmission electron microscopy revealed the anatomic compartments to which silica crystals were translocated over a 14 day post-exposure period. Particles were found in alveolar Type I cells, in interstitial cells and connective tissue, and within alveolar macrophages. Thirty-five to 65% of the *in situ* macrophages contained silica depending upon the time period studied. Approximately 10-20% of these silica-containing cells appeared necrotic, while only 1-4% of macrophages from unexposed animals showed ultrastructural alterations.

Using x-ray energy spectrometry, we compared the elemental content of lavaged macrophages from exposed and unexposed animals. This technique allowed us to determine the percentage of silica-exposed cells which exhibited silicon x-ray counts statistically significantly different ($p < .01$) from the non-specific background of the control (unexposed) population. The data showed that immediately after expsoure, 27% of the macrophages contained silica crystals. Twelve hours after exposure this percentage increased to 72%, and no significant differences in this level were detected until 42 days post-exposure at which time the percentage of silica positive cells returned to 28%. Additional evidence of ongoing particle clearance is shown where the percentage of cells with silicon x-ray counts greater than 3000 (cts/30 sec) peaks at 22% (12 hrs after exposure) and returns to 1% at 42 days.

We have studied the deposition and translocation of aerosolized silica crystals in the lungs of rats exposed for 3 hrs. Large numbers of particles